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Prepulse Inhibition Deficits only in Females with Obsessive Compulsive Disorder

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Abstract

Background—Deficits in sensorimotor gating have been hypothesized to underlie the inability to inhibit repetitive thoughts and behaviors. To test this hypothesis, this study assessed prepulse inhibition (PPI), a measure of sensorimotor gating, across three psychiatric disorders (obsessive-compulsive disorder [OCD], social anxiety disorder [SAD], and anorexia nervosa [AN]) whose clinical presentation includes repetitive thoughts and behaviors.

Methods—We tested acoustic PPI in unmedicated individuals with OCD (*n*=45), SAD (*n*=37), and AN (*n*=26), and compared their results to matched healthy volunteers (*n*=62). All participants completed a structured clinical interview and a clinical assessment of psychiatric symptom severity.

Results—Percent PPI was significantly diminished in females with OCD compared to healthy female volunteers (*p*=0.039). No other differences between healthy volunteers and participants

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Ethical Standards

All procedures contributing to this work comply with the ethical standards of New York State Psychiatric Institute's Institutional Review Board, the granting agency, and with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

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with disorders (male or female) were observed. Percent PPI was not correlated with severity of obsessions and compulsions, as measured by the Yale-Brown Obsessive Compulsive Scale.

Conclusions—This is the first study to assess PPI in participants with SAD or AN, and the largest study to assess PPI in participants with OCD. We found PPI deficits only in females with OCD, which suggests that the cortico-striato-pallido-thalamic and pontine circuitry (believed to underlie PPI) differs between males and females with OCD. Given that PPI deficits were only present in females with OCD and not related to repetitive thoughts and behaviors, our results do not support the hypothesis that sensorimotor gating deficits, as measured by PPI, underlie the inability to inhibit repetitive thoughts and behaviors in individuals with OCD, SAD, and AN.

Keywords

obsessive compulsive disorder; social anxiety disorder; eating disorders; anxiety; biological markers

Although constantly bombarded with stimuli, humans can filter out extraneous stimuli from awareness. This process, called ‘sensorimotor gating,’ relies upon cortico-striato-pallidal circuitry [1]. Sensorimotor gating deficits have been hypothesized to be related to the inability to inhibit repetitive thoughts and behaviors [2–4]. This paper reports results from the first study to assess sensorimotor gating deficits, using prepulse inhibition (PPI), across three psychiatric disorders (obsessive-compulsive disorder [OCD], social anxiety disorder [SAD], anorexia nervosa [AN]) whose clinical presentation includes repetitive thoughts and behaviors.

PPI is a well-validated measure of sensorimotor gating [1]. PPI refers to the normal dampening of the startle reflex when a startling auditory stimulus is preceded by a weaker sound [5]. In humans, it is measured by assessing the eyeblink component of the startle reflex following a loud startling tone (the pulse), preceded by a barely audible tone (the prepulse). The prepulse “gates” the startle reflex in response to the pulse. Rodent and human brain imaging studies indicate that PPI relies upon cortico-striato-pallido-thalamic and pontine circuitry, specifically the subpallidal efferents to the pedunculopontine nucleus, basal ganglia, thalamus, amygdala, hippocampus, and prefrontal cortex [6; 7].

PPI deficits have been demonstrated in several disorders, including OCD [2; 3; 8], schizophrenia [9; 10], and tic disorders [2; 11]. These disorders share dysfunction in cortico-striato-pallido-thalamic circuitry [4; 6] and the inability to inhibit repetitive thoughts (thought insertion in schizophrenia, obsessions in OCD) and repetitive behaviors (compulsions in OCD, tics in tic disorders). To our knowledge, no studies have evaluated PPI in a large, unmedicated sample across other psychiatric disorders whose clinical presentations also include repetitive thoughts and behaviors.

The current study evaluated sensorimotor gating using PPI across three psychiatric disorders: OCD, AN, and SAD, and healthy control volunteers (HC). This is the first study to assess PPI in SAD and AN, and the largest study to assess PPI in OCD. These disorders were selected because repetitive thoughts and behaviors occur in all three, although the thoughts and behaviors differ in degree, type, and content. Specifically, obsessions and

compulsions are the hallmark of OCD [12], SAD includes thoughts about evaluation and repetitive review of past social situations [13], and AN includes repetitive thoughts about food and rituals related to weight and shape [14]. Because of these shared clinical features, the relationship between these disorders has sparked debate in the field [15; 16]. We hypothesized that OCD participants would have PPI deficits compared to HC. We then explored whether AN or SAD also had PPI deficits compared to HC and whether PPI deficits were associated with the severity of repetitive thoughts and behaviors.

Methods and Materials

Study Description

The data in this paper come from a study conducted at the New York State Psychiatric Institute/Columbia University Medical Center. The overall aim was to determine whether well-validated neurobehavioral probes of different neural processes could help explain similarities and differences in phenotype across individuals with AN, OCD, and SAD. This paper describes the data collected on PPI as a measure of sensorimotor gating. The New York State Psychiatric Institute review board approved this study and participants provided written informed consent.

Participants

Adults aged 18–50 years with a principal diagnosis of OCD, SAD, or AN, and matched HC, were recruited via media and referrals from health professionals. Participants were free from psychiatric medication for at least four weeks (six weeks for fluoxetine), with the exception of one participant who took one dose of lorazepam two weeks prior to testing. Diagnoses were made by a psychiatrist, and confirmed by another clinician (MD or PhD) using a structured clinical interview [SCID-IV, 17]. HC had no lifetime Axis I psychiatric disorders. AN participants could have comorbid secondary OCD or SAD, due to the common co-occurrence of these disorders [18]. No other current comorbid Axis I disorders were permitted except for specific phobias or tic disorders. Individuals with AN were inpatients who were receiving treatment, and were required to have a body mass index (BMI) between 16.0 kg/m^2–18.5 kg/m^2 at the start of study procedures, to exclude participants with extreme starvation, due to its potential effects on performance.

Additional exclusion criteria included: 1) lifetime psychotic disorder, bipolar disorder, mental retardation, attention deficit hyperactivity disorder, or primary hoarding; 2) use of medication affecting the central nervous system, 3) hearing loss (measured by a hearing test performed bilaterally with tones of 500, 1000, 3000, and 6000 Hz at 35 dB); 4) habitual caffeine use with inability to refrain from caffeine for four hours prior to study without withdrawal symptoms; 5) habitual tobacco use (>five cigarettes per day) or inability to refrain from smoking for 24 hours prior to study without withdrawal symptoms; 6) medical or neurological problems that could interfere with study performance or interpretation; and 7) active suicidal ideation.

Females who were pregnant, nursing, postmenopausal, or using hormonal methods of birth control were excluded. All but three AN participants were amenorrheic. To reduce potential

confounds of hormonal status, the three menstruating AN participants were excluded from PPI analyses, and all other females were tested within the first week of their menstrual cycle.

Clinical Assessment

Clinician-administered measures were administered by a PhD-level psychologist. OCD severity was assessed with the Yale-Brown Obsessive Compulsive Scale [Y-BOCS, 19] which includes a checklist of possible obsessions and compulsions and subscales that measure severity of obsessions and of compulsions. SAD severity was assessed with the Liebowitz Social Anxiety Scale [LSAS, 20], which assesses fear and avoidance in situations involving social interaction or performance. The Y-BOCS and the LSAS have good psychometric properties [19; 21; 22].

Self-report measures included the Eating Disorder Examination Questionnaire [EDE-Q, 23], the State Trait Anxiety Inventory–Trait Subscale [STAI-Trait, 24], and the Quick Inventory of Depressive Symptomatology [QIDS, 25]. The EDE-Q assesses the severity of anorexia and bulimia, the STAI-Trait is a measure of trait anxiety, and the QIDS assesses the severity of depressive symptoms. All have good psychometric properties [24–26]. Participants completed the North American Adult Reading Test [NAART, 27] to estimate IQ.

Measurement of PPI and Habituation

An EMG startle system (EMG-SR-LAB, San Diego Instruments, San Diego, CA, USA) recorded participants' orbicularis oculi electromyogram (EMG) responses following loud startling noises. For each participant, two small silver/silver chloride electrodes were placed over the orbicularis oculi muscle (below and to the side of the right eye). Electrode resistance was <10kΩ. Participants were instructed to sit comfortably, keep their eyes open, and look straight ahead at a fixation point on the wall. They were informed that they would hear white noise and loud bursts of noise through binaural headphones. Once the headphones were in place, participants were presented with a 4 min acclimation period of 70 dB broad-band noise, which continued as background noise throughout the PPI session.

The PPI session included a total of 71 trials. Each session included a no-stimulus trial (70 dB), a pulse alone (PA) trial, and three prepulse+pulse (PP) trials, pseudorandomly presented in five 12-trial blocks. Each session began with a single PA trial, and included five PA trials at the beginning and end of the session to assess habituation (71 trials total=5 PA [Habituation1]+1PA+60[5 x 12] block trials+5 PA [Habituation2]). In PA trials, a 116 dB white noise burst was presented for 40 ms over the 70 dB background noise. In PP trials, short (20 ms) white noise bursts of 74, 78, or 86 dB were presented, with the 116 dB white noise burst occurring 120 ms after the onset of the prepulse. Intertrial intervals varied from 10 to 20 s (average=15 s). Sampling rate was 1000 Hz, and amplifier gain was set at 0.50. EMG recordings started at the onset of the startle stimulus and continued for 250 ms.

Data Processing

As in prior studies [2; 9], data were filtered with a band-pass filter (100–1000 Hz) and smoothed with a 10 ms rolling average. Spontaneous and voluntary blinks were excluded, following criteria described in Braff et al. [9]. Using SRRED software (San Diego

Instruments, San Diego CA), peak startle magnitude and peak latency were calculated. Twenty-two (11 HC, 6 OCD, 5 SAD) participants were insufficient startlers (average amplitude of the pulse alone trials was <3 times the average amplitude of the no-stimulus trials), and were excluded from analyses.

In those who startled, individual trials were excluded from analyses if they had pulse onset-to-peak latency greater than 100 msec (suggesting eyeblink was voluntary or spontaneous, and not linked to stimulus) or if they had baseline shifts due to excessive background noise or voluntary blinks closely preceding the startle stimulus. Peak latency was operationalized as the maximal amplitude occurring within 100 msec after startle stimulus. A standard computer algorithm determined maximal amplitude (obtained from SRRED).

Individual sessions were scored by S.L., with >50% of the files also verified by V.R. to ensure standardized scoring. S.L. and V.R. had no contact with participants, and were blind to diagnosis.

Outcome Measures

The primary outcome measure, percent PPI, was calculated as the percent change in the mean startle magnitude from the PA trials to the mean startle magnitude at each of the three PP trial intensity levels (74, 78, or 86 dB). Specifically, the percent PPI was calculated for each person at each dB level as [(mean(PA)-mean(PPdB))/mean(PA)]x100, where mean(PA) indicates the mean over the within-person PA trials, and mean(PPdB) indicates the mean over the within-person PP trials at a specific dB level. Habituation was assessed by comparing mean startle of the first five PA trials with the mean startle of the last five PA trials [(mean(pretest PA) – mean(posttest PA))/mean(pretest PA)]x100. The 10 PA trials used to assess habituation were not included in PPI calculations.

Statistical Analysis

To test for group differences in demographic and clinical measures, one-way ANOVAs were used for continuous measures and Fisher's exact tests for categorical variables. To test for habituation, a repeated measures ANOVA modelled the first and last 5 PA trials on block (beginning vs. end), diagnostic group, and their interaction. To test for potential differences in mean startle reactivity, all but the 5 first and last PA trials (13 trials) were averaged and one-way ANOVAs were used to assess differences between groups.. To assess if percent PPI differed by diagnostic group, a general linear mixed model was used including a diagnostic group factor (HC, OCD, SAD, AN), a PP dB intensity factor (74, 78, 86), a group*intensity factor, and a random intercept to control for repeated measures within individuals. To test whether sex moderated results, the model also included sex, sex*group, sex*intensity, and sex*group*intensity factors. Analyses were repeated controlling for race/ethnicity (because this differed between groups); results did not change when including race/ethnicity as a covariate, so it is not included in the final model. Statistical contrasts testing the mean percent PPI differences between each diagnostic group (OCD, SAD, AN) and the HC group were formed from results of the model. Statistical significance was defined as $p<.05$, and no corrections were made for multiple testing. Effect sizes (ES) for group

differences were calculated by dividing the mean percent PPI differences by the standard deviation of percent PPI in the overall sample.

To test whether percent PPI correlated with repetitive thoughts and behaviors, Spearman correlations (robust to outliers) were used to estimate associations between the mean percent PPI (aggregated across dB levels) and Y-BOCS Total Score, Y-BOCS Obsession Subscale, and Y-BOCS Compulsion Subscale. Correlations were calculated across the full sample, within the OCD group, and within the female OCD group. Analyses were performed using SAS version 9.3.

Results

Demographics and clinical characteristics

Of the 217 individuals who consented, seven did not return after consenting, and 10 were found to meet exclusion criteria after consenting. Of the 200 individuals who were eligible and participated in the parent study (75 HC, 51 OCD, 44 SAD, and 30 AN), 170 had analyzable PPI data and are included in the following analyses. The other 30 participants were excluded for various reasons: insufficient startle (n=22), experimenter or technical error (n=4), participant was a menstruating female with anorexia (n=3), participant was a male with AN (n=1). Participants with analyzable PPI data included 62 HC, 45 OCD, 37 SAD, and 26 AN (of whom 3 had comorbid OCD and 4 had comorbid SAD). Nine participants had current secondary specific phobia (3 OCD participants, 3 AN participants, and 3 SAD participants) and 6 participants had secondary tic disorder (4 OCD participants and 2 AN participants). Analyses were re-run two ways: 1) excluding AN participants with comorbid OCD or SAD; and 2) excluding participants with comorbid specific phobia or tic disorders. The general pattern of results did not change, so all participants with analyzable data are included in results presented below.

Demographic and clinical characteristics are shown in Table 1. Most participants had never taken psychiatric medications. Groups did not differ in age, IQ (as measured by the NAART), or years of education (all p 0.246; see Table 1). Groups differed in sex (p<0.001) and race/ethnicity (p=0.004), due to the fact that AN participants were all white females. Excluding the AN participants, diagnostic groups did not differ in sex or race/ethnicity (both $p \leq 0.313$).

As expected, subjects differed on clinical characteristics (Table 1): the OCD group reported higher levels of OCD symptoms (as measured by the Y-BOCS) than each of the other groups (all p<0.001); the SAD group reported higher levels of social anxiety symptoms (as measured by the LSAS) than each of the other groups (all p<0.001); and the AN group reported higher levels of eating disorder symptoms (as measured by the EDE-Q) than each of the other groups (all p <0.001). The AN group reported the highest levels of anxiety (assessed by the STAI Trait) and depression (assessed by the QIDS; all p 0.016 for comparisons with each of the other groups). The HC group reported the lowest levels of anxiety and depression on the STAI Trait and the QIDS (all p<.001 for comparisons with each of the other groups).

PPI Results

Habituation and startle reactivity—As expected, all groups habituated over the course of the PPI session, with a significant effect for block (beginning vs end of session: $F(1,169)=326.17$; $p<0.001$). Neither the group effect, nor the group*block interaction were found to be significant (group: $F(3,166)=1.37$; $p=0.255$; interaction: $F(3,166)=0.64$; $p=0.590$), indicating no difference between groups in habituation (see Figure 1). In PA trials that were used to calculate PPI, groups also did not differ in mean startle reactivity (mean [standard deviation]: HC: 409.82 [286.08]; AN: 400.27 [274.41]; OCD: 464.41 [366.92]; SAD: 423.47 [285.51]; $F(3,166)=0.35$, $p=0.788$).

PPI of the Acoustic Startle Response—Means and standard deviations of Percent PPI for each group, decibel level, and sex are shown in Table 2. As expected, the percent PPI increased as PP Intensity increased ($F(2,338)=157.78$, $p<0.001$). The mean percent PPI for the HC group was 38.44% (standard error, SE=3.58%), indicating that compared to the PA condition, HC participants decreased their startle response by 38.44% when a prepulse was presented (averaging over the three PP dB intensity levels). Compared to the HC group, the adjusted mean percent PPI for the OCD group was lower but did not reach statistical significance (30.73%, SE=4.20%; $p=0.112$), and represented a small effect size of 0.28. However, the diagnostic group*sex interaction in the model was significant ($F(2,330)=3.16$, $p=0.044$; see Figure 2). Post-hoc contrasts found that OCD females had significantly lower percent PPI than HC females (OCD: 19.70%, SE=6.00%; HC: 35.64%, SE=4.79%; $p=0.039$), and the effect size was moderate (ES=0.57). There was no significant difference between OCD and HC males (OCD: 40.39%, SE=5.61%; HC: 41.63%, SE=5.10%, $p=0.870$, ES=0.04). Although the diagnostic group*dB intensity level interaction was not significant ($F(6,330)=0.88$, $p=0.506$), we present the HC vs. OCD contrasts for each dB level, so that our findings can be compared with past literature (Table 2).

The mean percent PPI for the HC group did not differ significantly from the SAD group (34.34%; SE=4.63%; ES= 0.18, $p=0.363$) or the AN group (28.05%; SE=5.39%; ES= 0.27, $p=0.293$). Percent PPI of the AN group was compared to percent PPI of females in the HC group (35.64%, SE=4.79%). This pattern did not change when analyses excluded the three AN participants with comorbid OCD. There was a significant difference between females with OCD and SAD (38.51%; SE=5.86, ES=0.55, $p=0.026$), but not between females with AN and OCD (ES=0.22, $p=0.301$); however, these analyses should be interpreted cautiously, given that they were not planned analyses. Post-hoc tests found no significant percent PPI differences between SAD and HC groups in males or females (p 0.126). To demonstrate inter-subject variability, the mean percent PPI for each participant is presented in Figure 3a (full sample), 3b (males only), and 3c (females only).

Relationships between Y-BOCS and PPI—The mean percent PPI was not correlated with Y-BOCS Total Score, Y-BOCS Obsession Subscale, or the Y-BOCS Compulsion Subscale in the full sample, within the OCD group, or within the female OCD group (all p 0.199).

Discussion

In this study, the largest to measure PPI in participants with OCD and the first to assess PPI in participants with AN and SAD, we found that PPI was diminished in females with OCD compared to healthy females. No other differences between HC and participants with disorders (male or female) emerged. Further, we found no association between percent PPI and severity of repetitive thoughts and behaviors (as measured by the Y-BOCS).

The finding that female OCD participants had diminished PPI compared to HC replicates previous findings in smaller samples showing decreased PPI in OCD participants [2; 3; 8], and provides some support for sensorimotor gating deficits in individuals with OCD. Contrary to prior reports, we found PPI deficits only in female (and not male) OCD participants. On the other hand, re-analysis of the data from Ahmari *et al.* [2] indicated that the effect size comparing females with OCD to HC was larger than that seen in males [28]. Among other PPI studies in OCD, one [3] did not evaluate sex effects, and another [29] did not find a sex by diagnosis interaction, but small sample size (11 OCD participants) and failure to standardize menstrual cycle phase limits interpretation of these results. Finally, the only published study that did not find PPI deficits in OCD [30] had a much smaller proportion of females (28% of OCD participants were female, compared to 45–50% female in other studies). Why females with OCD would be more likely to have PPI deficits than males with OCD is not clear, but could reflect gender differences in the neurobiology of OCD and deserves further study.

We also found no correlation between PPI deficits and the severity of repetitive thoughts and behaviors (as measured by the Y-BOCS). This contradicts the hypothesis that PPI deficits are functionally linked to the inability to inhibit repetitive thoughts and behaviors. In fact, only one of four prior PPI studies in OCD found a significant relationship between PPI and Y-BOCS scores[3]. Thus, across the majority of studies, PPI deficits are not strongly associated with the severity of repetitive thoughts and behaviors, at least as measured by the Y-BOCS. Because PPI is only one measure of sensorimotor gating, it is possible that other measures of sensory gating [e.g., paired-click paradigm, 31] are more strongly linked with the severity of repetitive thoughts and behaviors.

There were no significant differences in percent PPI either in AN compared to the HC group, or in SAD compared to the HC group. Inspection of means suggests that, similar to the OCD females, the (all female) AN group had lower PPI than the SAD and HC groups. Additionally, while not a planned analysis, we found that percent PPI was significantly lower in females with OCD compared to females with SAD, but there was no difference in percent PPI between between females with AN and OCD. An unpublished study [32] found reduced percent PPI among participants who had recovered from an eating disorder (10 AN and 9 bulimia nervosa) compared with HC, though the small sample size limits interpretation of findings. Individuals with AN and OCD show abnormalities in frontostriatal circuits and share some aspects of phenomenology [33]. Future studies should further evaluate sensorimotor gating in participants with current eating disorders.

This study has several strengths. Specifically, the sample was large and we evaluated PPI using identical methods across three psychiatric disorders. Additionally, all participants were free of psychiatric medication [particularly important, given that psychiatric medication can affect PPI; 34; 35]. All females were tested within the first week of their menstrual cycle to control for menstrual effects [36]. Further, the sample was relatively free of comorbidity.

Some limitations are worth noting. First, the AN sample was small and differed demographically from the other groups. Second, at the time of data collection, the only validated measure of repetitive thoughts and behaviors was the Y-BOCS, which was developed specifically for OCD and may not be an ideal transdiagnostic measure. This concern highlights the need for validation of newer, transdiagnostic measures (e.g., the APA Repetitive Thoughts and Behavior Scale [adapted from the Florida Obsessive-Compulsive Inventory Severity Scale, 37]

In summary, we found sensorimotor deficits, as measured by PPI, only in female participants with OCD. Assuming PPI deficits reflect dysfunction in cortico-striato-pallido-thalamic and pontine circuitry, these findings are consistent with the notion that this circuitry may differ between males and females with OCD. Given that PPI deficits were only present in females with OCD, and were not related to the severity of repetitive thoughts and behaviors, our results do not support the hypothesis that sensorimotor gating deficits, as measured by PPI, underlie the inability to inhibit repetitive thoughts and behaviors, either within OCD or across disorders.

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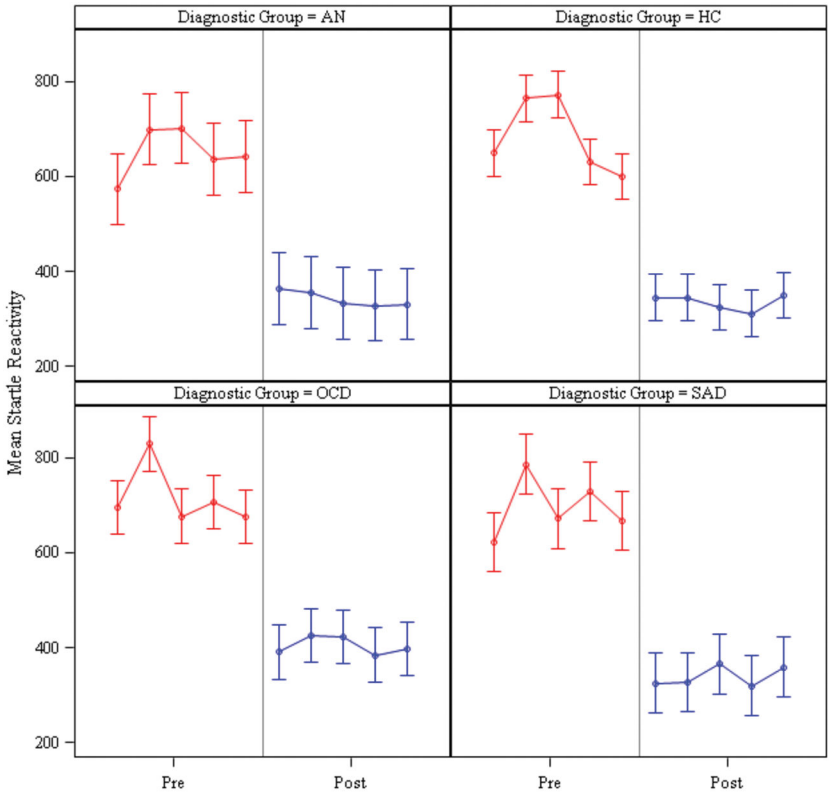

Figure 1.

Mean Startle Reactivity during Habituation Trials

Note. HC=Healthy Control Group, OCD=Obsessive Compulsive Disorder Group, SAD=Social Anxiety Disorder Group, AN=Anorexia Nervosa Group, Pre=first five Pulse Alone trials, Post=last five Pulse Alone trials.

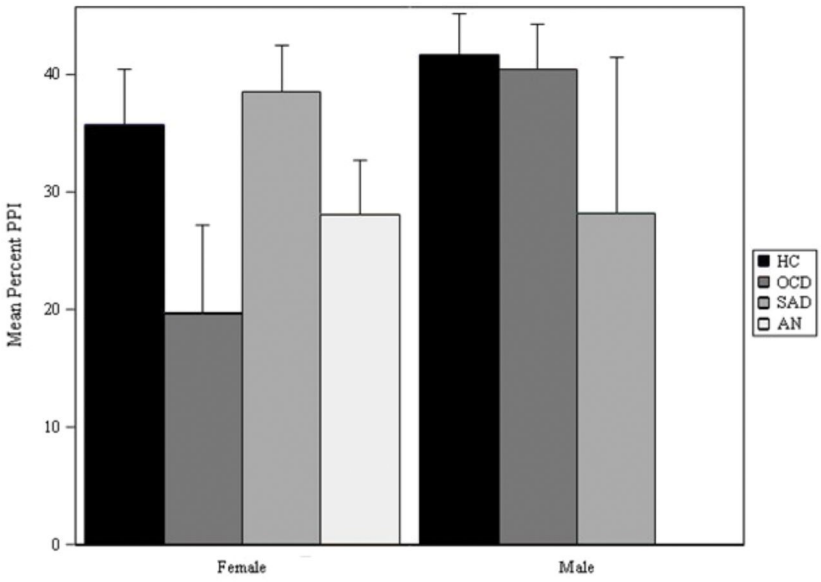

Figure 2.

Mean Percent PPI by Diagnostic Group and Sex

Note. HC=Healthy Control Group, OCD=Obsessive Compulsive Disorder Group, SAD=Social Anxiety Disorder Group, AN=Anorexia Nervosa Group

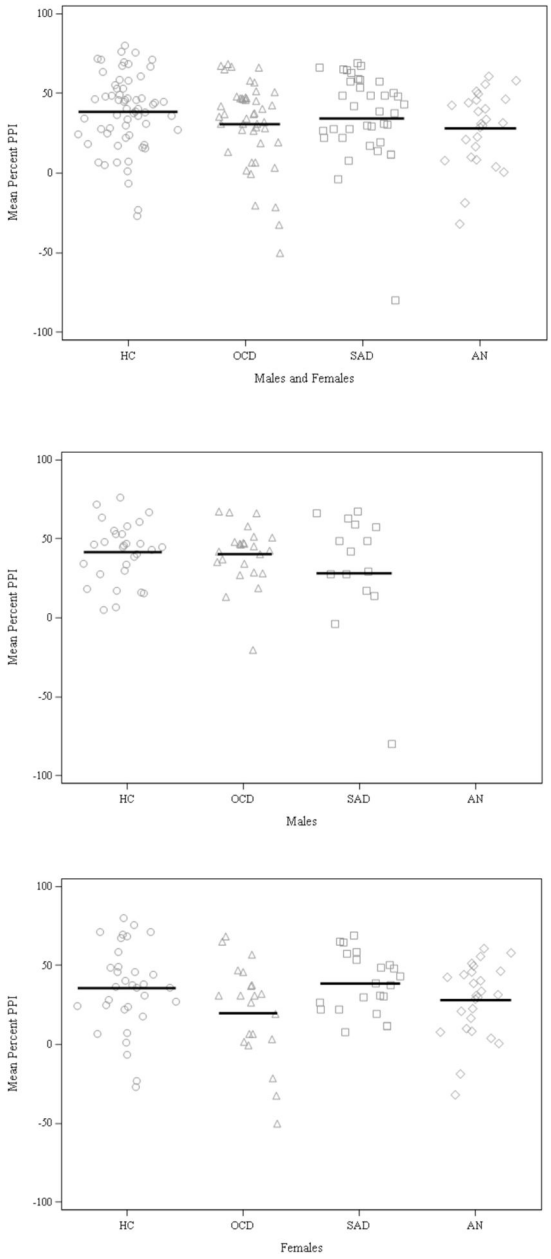

Figure 3.

Figure 3a. Mean Percent PPI for each Participant by Diagnostic Group, Males and Females Figure 3b. Mean Percent PPI for each Participant by Diagnostic Group, Males Only Figure 3c. Mean Percent PPI for each Participant by Diagnostic Group, Females Only

Note. HC=Healthy Control Group, OCD=Obsessive Compulsive Disorder Group, SAD=Social Anxiety Disorder Group, AN=Anorexia Nervosa Group

Table 1

Demographics and Clinical Characteristics

	HC *n*=62Mean (SD)	OCD *n*=45 Mean (SD)	AN *n*=26 Mean (SD)	SAD *n*=37 Mean (SD)	*p*-value[a,b]
Age	27.60 (6.50)	28.80 (5.89)	26.93 (7.67)	28.54 (6.66)	0.611
Sex (n, % Female)	33 (53%)	21 (47%)	26 (100%)	22 (59%)	< .001
Race/Ethnicity (n, %)					0.004
Non-Hispanic White	37 (60%)	26 (58%)	25 (96%)	14 (38%)	
Hispanic White	11 (18%)	8 (18%)	1 (4%)	10 (27%)	
Asian	3 (5%)	3 (7%)	0	4 (11%)	
Black	10 (16%)	7(16%)	0	5 (14%)	
Other	1 (2%)	1 (2%)	0	4 (11%)	
Years of Education	15.79 (2.06)	15.40 (1.99)	14.85 (2.05)	15.57 (1.92)	0.246
Estimated IQ (NAART)	110.24 (9.05)	110.04 (8.72)	108.85 (8.49)	110.92 (7.86)	0.826
Age of Onset	-	13.36 (5.57)	16.04 (2.40)	11.20 (5.90)	0.003
EDE-Q-Global	0.54 (0.63)	0.93 (0.93)	3.52 (1.60)	0.89 (0.92)	<.001
LSAS-Total	12.42 (8.01)	23.04 (17.03)	47.81 (20.14)	75.00(19.32)	<.001
QIDS-Total	2.40 (2.00)	5.58 (4.28)	12.58 (4.54)	6.00 (3.68)	<.001
STAI-Trait	31.65 (4.99)	43.31 (11.19)	54.18 (9.31)	48.92 (8.43)	<.001
Y-BOCS-Total	0.27 (1.03)	24.98 (3.46)	10.42 (10.61)	2.91 (5.35)	<.001

Note. HC=Healthy Control Group, AN=Anorexia Nervosa Group, OCD=Obsessive Compulsive Disorder Group, SAD=Social Anxiety Disorder Group, NAART=North American Adult Reading Task, EDE-Q=Eating Disorder Examination Questionnaire, LSAS=Liebowitz Social Anxiety Scale, QIDS=Quick Inventory of Depressive Symptomatology, STAI=State Trait Anxiety Inventory, Y-BOCS=Yale-Brown Obsessive Compulsive Scale

[a] *p*-value for ANOVA (Age, NAART, Education, Y-BOCS, LSAS, EDE-Q, STAI, QIDS) and Fisher's exact test (sex, race/ethnicity) for any group differences.

[b] *p*-value for testing group differences excluding AN, who are all white females, is p=0.529 for sex, and 0.313 for race/ethnicity.

Table 2

Percent PPI by Diagnosis, dB intensity, and Sex (Mean (SD))

Females	**HC *n*=33**	**OCD *n*=21**	**AN *n*=26**	**SAD *n*=22**
74 dB	19.97 (30.74)	14.14 (23.84)	13.57 (24.15)	24.30 (16.03)
78 dB	36.32 (30.06)	**17.94 (34.91)**[a]	27.29 (25.57)	37.74 (20.46)
86 dB	50.63 (27.87)	**27.00 (48.71)**[b]	43.30 (26.60)	53.49 (26.07)
Overall	35.64 (27.15)	**19.70 (34.41)**[c]	28.05 (23.37)	38.51 (18.64)
Males	**HC** *n*=29	**OCD** *n*=24	**AN** *n*=0	**SAD** *n*=15
74 dB	30.25 (17.67)	27.07 (18.89)	-	15.07 (51.33)
78 dB	40.85 (22.91)	39.45 (22.08)	-	29.58 (45.65)
86 dB	53.78 (21.51)	54.64 (22.43)	-	40.02 (60.05)
Overall	41.63 (18.83)	40.39 (19.06)		28.22 (51.19)
Combined	**HC** *n*=62	**OCD** *n*=45	**AN** *n*=0	**SAD** *n*=37
74 dB	24.78 (25.81)	21.04 (22.08)	-	20.55 (34.58)
78 dB	38.44 (26.84)	29.42 (30.44)	-	34.43 (32.73)
86 dB	52.11 (24.95)	41.74 (39.19)	-	48.03 (42.94)
Overall	38.44 (23.63)	30.73 (28.93)		34.34 (35.33)

Note. HC=Healthy Control Group, OCD=Obsessive Compulsive Disorder Group, AN=Anorexia Nervosa Group, SAD=Social Anxiety Disorder Group.

[a] Significantly different from HC Females at 78 dB (p=0.027)

[b] Significantly different from HC Females at 86 dB (p=0.005)

[c] Significantly different from HC Females (p=0.039)